What is claimed is:

1. Use of an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of an effector molecule to a DC-SIGN receptor for the preparation of a medicament for preventing or treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of the effector molecule to the DC-SIGN receptor of the mammal to be treated.

2. Use of an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of an effector molecule to a DC-SIGN receptor for the preparation of a medicament for preventing or treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of the effector molecule to the DC-SIGN receptor of the mammal to be treated.

3. The use of claim 2, wherein the DC-SIGN blocker is a blocking derivative of the effector molecule.

4. The use of claim 2, wherein the DC-SIGN blocker is an antibody.

5. The use of claim 4, wherein the antibody specifically binds DC-SIGN.

6. The use of claim 4, wherein the antibody specifically binds the effector molecule.

7. The use of claim 2, wherein the DC-SIGN blocker is a mannosylated molecule that binds to a DC-SIGN receptor.

8. The use of claim 7, wherein the mannosylated molecule is mannan.

9. Use of an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of a viral effector molecule to a DC-SIGN receptor for the preparation of a medicament for preventing or treating a viral infection of a mammal, wherein the viral infection is mediated at least in part by the binding of the viral effector molecule to the DC-SIGN receptor of the mammal to be treated.

10. Use of an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of a viral effector molecule to a DC-SIGN receptor for the preparation of a medicament for preventing or treating a viral infection of a

mammal, wherein the viral infection is mediated at least in part by the binding of the viral effector molecule to the DC-SIGN receptor of the mammal to be treated.

11. The use of claim 10, wherein the viral effector molecule is a molecular constituent of the viral envelope.

12. The use of claim 11, wherein the molecular constituent of the viral envelope is an envelope glycoprotein.

13. The use of claim 10, wherein the DC-SIGN blocker comprises a binding moiety of the viral effector molecule.

14. The use of claim 12, wherein the DC-SIGN blocker comprises a binding moiety of the envelope glycoprotein.

15. The use of claim 10, wherein the DC-SIGN blocker is an antibody.

16. The use of claim 15, wherein the antibody is a monoclonal antibody.

17. The use of claim 16, wherein the mammal is a human and the monoclonal antibody is humanized.

18. The use of claim 15, wherein the antibody specifically binds DC-SIGN.

19. The use of claim 15, wherein the antibody specifically binds the viral effector molecule.

20. The use of claim 19, wherein the antibody specifically binds the binding moiety of the viral effector molecule.

21. The use of claim 10, wherein the DC-SIGN blocker is a mannosylated molecule that binds to a DC-SIGN receptor.

22. The use of claim 21, wherein the mannosylated molecule is mannan.

23. The use of claim 10, wherein the viral infection is a *Flaviviridae* virus infection and the viral effector molecule is a *Flaviviridae* effector molecule.

24. The use of claim 23, wherein the mammal is a human.

25. The use of claim 23, wherein the *Flaviviridae* viral infection is a Dengue virus infection and the *Flaviviridae* effector molecule is a Dengue effector molecule.

26. The use of claim 25, wherein the Dengue virus effector molecule is a molecular constituent of the Dengue virus envelope.

27. The use of claim 26, wherein the molecular constituent of the Dengue virus envelope is a Dengue virus envelope glycoprotein.

28. The use of claim 27, wherein the Dengue virus envelope glycoprotein is Dengue virus E glycoprotein.

29. The use of claim 25, wherein the DC-SIGN blocker comprises a binding moiety of the Dengue virus effector molecule.

30. The use of claim 28, wherein the DC-SIGN blocker comprises a binding moiety of the Dengue virus E glycoprotein.

31. The use of claim 30, wherein the DC-SIGN blocker is a recombinantly produced protein.

32. The use of claim 25, wherein the DC-SIGN blocker is an antibody.

33. The use of claim 32, wherein the antibody is a monoclonal antibody.

34. The use of claim 33, wherein the monoclonal antibody is humanized.

35. The use of claim 32, wherein the antibody specifically binds DC-SIGN.

36. The use of claim 32, wherein the antibody specifically binds the Dengue virus effector molecule.

37. The use of claim 36, wherein the Dengue virus effector molecule is Dengue virus E glycoprotein.

38. Use of an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of HIV or SIV to a DC-SIGN receptor present on dendritic cells of a human or a simian for the preparation of a medicament for preventing or treating an HIV or a SIV infection of said human or said simian.

39. Use of an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of HIV or SIV to a DC-SIGN receptor present on dendritic

cells of a human or a simian for the preparation of a medicament for preventing or treating an HIV or a SIV infection of said human or said simian.

40. The use of claim 39, wherein the DC-SIGN blocker comprises a binding moiety of the Dengue virus E glycoprotein.

41. The use of claim 39, wherein an HIV infection of a human is prevented or treated.

42. Use of an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of ICAM-3 present on T cells of a mammal with DC-SIGN receptor present on dendritic cells of the mammal for the preparation of a medicament for preventing or treating inflammation in said mammal caused by specific binding of ICAM-3 present on T cells of the mammal with DC-SIGN receptor present on dendritic cells of the mammal.

43. Use of an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of ICAM-3 present on T cells of a mammal with DC-SIGN receptor present on dendritic cells of the mammal for the preparation of a medicament for preventing or treating inflammation in said mammal caused by specific binding of ICAM-3 present on T cells of the mammal with DC-SIGN receptor present on dendritic cells of the mammal.

44. The use of claim 43, wherein the DC-SIGN blocker comprises a binding moiety of the Dengue virus E glycoprotein.

45. The use of claim 43, wherein the mammal is a human.

46. A pharmaceutical composition comprising:

a) A DC-SIGN blocker, and

b) at least one pharmaceutically acceptable excipient;

wherein the DC-SIGN blocker is present in the composition at an achievable therapeutic concentration.

47. The pharmaceutical composition of claim 46, wherein the DC-SIGN blocker is a derivative of a viral effector molecule.

48. The pharmaceutical composition of claim 46, wherein the DC-SIGN blocker comprises the binding moiety of a Dengue virus effector molecule.

49. The pharmaceutical composition of claim 48, wherein the Dengue virus effector molecule is Dengue virus E glycoprotein.

50. The pharmaceutical composition of claim 46, wherein the DC-SIGN blocker is an antibody.

51. The pharmaceutical composition of claim 50, wherein the antibody is a monoclonal antibody.

52. The pharmaceutical composition of claim 51, wherein the monoclonal antibody is humanized.

53. The pharmaceutical composition of claim 50, wherein the antibody specifically binds DC-SIGN.

54. The pharmaceutical composition of claim 50, wherein the antibody specifically binds the viral effector molecule.

55. The pharmaceutical composition of claim 54, wherein the antibody specifically binds the binding moiety of the viral effector molecule.

56. A method of identifying a DC-SIGN modulator, wherein the method comprises:

a) determining a baseline binding value by:

i. providing cultured cells comprising a DC-SIGN receptor;

ii. exposing the cultured cells to a marked viral effector molecule binding moiety for a period of time sufficient to allow binding equilibrium to be reached; and

iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a baseline binding value;

b) determining a test substance binding value by:

i. providing cultured cells comprising a DC-SIGN receptor;

ii. exposing the cultured cells to a marked viral effector molecule binding moiety in the presence of a test substance for a period of time sufficient to allow binding equilibrium to be reached; and

iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a test substance binding value; and

c) determining a test substance binding modulation value for the test substance by dividing the test substance binding value by the baseline binding value,

wherein a test substance binding modulation value representing an about 95% modulation of binding of the viral effector molecule to dendritic cells by the test substance, indicates that the test substance is a substance that substantially modulates the binding of a viral effector molecule to the DC-SIGN receptor.

57. A method of identifying a DC-SIGN blocker, wherein the method comprises:

a) determining a baseline binding value by:

i. providing cultured cells comprising a DC-SIGN receptor;

ii. exposing the cultured cells to a marked viral effector molecule binding moiety for a period of time sufficient to allow binding equilibrium to be reached; and

iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a baseline binding value;

b) determining a test substance binding value by:

i. providing cultured cells comprising a DC-SIGN receptor;

ii. exposing the cultured cells to a marked viral effector molecule binding moiety in the presence of a test substance for a period of time sufficient to allow binding equilibrium to be reached; and

iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a test substance binding value; and

c) determining a test substance binding inhibition value for the test substance by dividing the test substance binding value by the baseline binding value,

wherein a test substance binding inhibition value representing an about 95% inhibition of binding of the viral effector molecule to dendritic cells by the

test substance, indicates that the test substance is a substance that substantially inhibits the binding of a viral effector molecule to the DC-SIGN receptor.

58. The method of claim 57 wherein the cultured cells are DC.

59. The method of claim 57, wherein the cultured cells are THP-1 cells.

60. The method of claim 57, wherein the viral effector molecule is a Dengue virus effector molecule.

61. The method of claim 60, wherein the Dengue virus effector molecule is Dengue virus E glycoprotein.

62. An isolated DC-SIGN blocker identified by the method of claim 57.

63. A method of targeting a subject molecule to a cell expressing a DC-SIGN receptor by exposing the cell to a targeting complex, wherein the targeting complex comprises a subject molecule and a DC-SIGN blocker, wherein the exposure is under conditions which allow the DC-SIGN blocker to bind to DC-SIGN on the cell expressing the DC-SIGN receptor, thereby targeting the subject molecule to the cell expressing a DC-SIGN receptor.

64. The method of claim 63, wherein the DC-SIGN blocker is an antibody.

65. The method of claim 64, wherein the antibody is a monoclonal antibody.

66. The method of claim 63, wherein the subject molecule is a protein.

67. The method of claim 63, wherein the subject molecule is an antibody.

68. The method of claim 63, wherein the subject molecule is labeled.

69. The method of claim 63, wherein the exposure occurs *in vivo*.

70. The method of claim 63, wherein the exposure occurs *in vitro*.

71. A pharmaceutical composition comprising:

a) A DC-SIGN modulator, and

b) at least one pharmaceutically acceptable excipient;

wherein the DC-SIGN modulator is present in the composition at an achievable therapeutic concentration.